EXHIBIT 5

FOUNDATION OVERVIEW

Founded in 1994, the Angiogenesis Foundation is the world's first 501(c)(3) nonprofit organization dedicated to conquering disease using a new approach based on angiogenesis, the growth of new capillary blood vessels in the body. We have identified angiogenesis as the "common denominator" in society's most feared diseases. Our focus on this underlying process of many different diseases makes our approach as a medical organization unique. The Foundation is the recognized, expert voice and champion of this new field of medicine.

Based in Cambridge, Massachusetts, the Angiogenesis Foundation's goal is to help people lead healthier, longer lives by restoring balance to blood vessel growth. Through research, education, and advocacy — with patients, physicians, researchers, industry, payers, and government — we enable patients to gain access to safe and effective treatments coming from the angiogenesis field for cancer, blinding diseases, wounds, and many other serious diseases.

Working with an international advisory board and a faculty of medical research experts, we are speeding the progress of modern medicine, teaching doctors how to adopt 21st century treatments as they emerge, and laying the groundwork for major, future breakthroughs. Because angiogenesis plays a role in so many different diseases, the Foundation 'cross-fertilizes' knowledge from one disease research area to another, so that medical progress for many diseases can be catalyzed at once.

As a scientific organization, the Angiogenesis Foundation is independent of any individual, institution, or commercial entity. We are committed to helping people around the world benefit from the full promise of angiogenesis-based medicine, and to make life-, limb-, and vision-saving treatments available to everyone in need.

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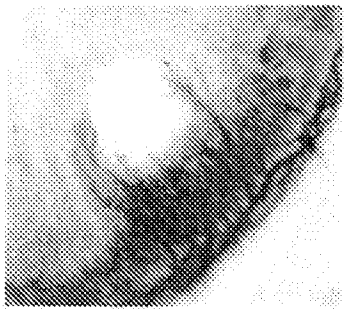


Angiogenesis, new capillary blood vessel growth, as seen by researchers in the laboratory.

Understanding Angiogenesis

Angiogenesis (angio'gen'esis) -- the growth of new blood vessels -- is an important natural process occurring in the body, both in health and in disease.

The Body's Control of Angiogenesis

Angiogenesis occurs in the healthy body for healing wounds and for restoring blood flow to tissues after injury or insult. In females, angiogenesis also occurs during the monthly reproductive cycle (to rebuild the uterus lining, to mature the egg during ovulation) and during pregnancy (to build the placenta, the circulation between mother and fetus).

The healthy body controls angiogenesis through a series of "on" and "off" switches:

- The main "on" switches are known as angiogenesis-stimulating growth factors
- The main "off switches" are known as angiogenesis inhibitors

When angiogenic growth factors are produced in excess of angiogenesis inhibitors, the balance is tipped in favor of blood vessel growth. When inhibitors are present in excess of stimulators, angiogenesis is stopped. The normal, healthy body maintains a perfect balance of angiogenesis modulators. In general, angiogenesis is "turned off" by the production of more inhibitors than stimulators.

Known Angiogenic Growth Factors

Angiogenin
Angiopoietin-1
Del-1
Fibroblast growth factors: acidic (aFGF) and basic (bFGF)
Follistatin
Granulocyte colony-stimulating factor (G-CSF)
Hepatocyte growth factor (HGF) /scatter factor (SF)
Interleukin-8 (IL-8)
Leptin
Midkine
Placental growth factor
Platelet-derived endothelial cell growth factor (PD-ECGF)
Platelet-derived growth factor-BB (PDGF-BB)
Pleiotrophin (PTN)
Progranulin
Proliferin
Transforming growth factor-alpha (TGF-alpha)
Transforming growth factor-beta (TGF-beta)
Tumor necrosis factor-alpha (TNF-alpha)
Vascular endothelial growth factor (VEGF)/vascular permeability factor (VPF)

Known Angiogenesis Inhibitors

Angioarrestin
Angiostatin (plasminogen fragment)
Antiangiogenic antithrombin III

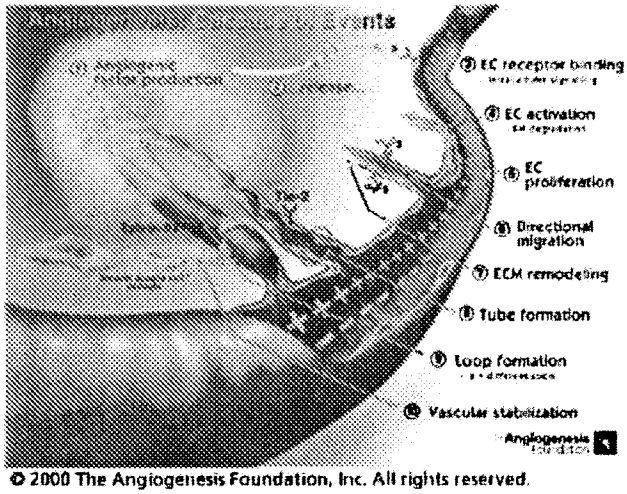
being developed to treat these conditions.

Angiogenesis is a disease common denominator

Angiogenesis, the growth of new blood vessels, is a "common denominator" shared by diseases affecting more than one billion people worldwide. This includes all cancers, cardiovascular disease, blindness, arthritis, complications of AIDS, diabetes, Alzheimer's disease, and more than 70 other major health conditions affecting children and adults in developed and developing nations. Our vision is that angiogenesis-based therapies are a unifying approach to disease and will have the same impact in the 21st century that antibiotics had in the 20th century.

The Angiogenesis Process: How Do New Blood Vessels Grow?

The process of angiogenesis occurs as an orderly series of events:



1. Diseased or injured tissues produce and release angiogenic growth factors (proteins) that diffuse into the nearby tissues.
2. The angiogenic growth factors bind to specific receptors located on the endothelial cells (EC) of nearby preexisting blood vessels.
3. Once growth factors bind to their receptors, the endothelial cells become activated. Signals are sent from the cell's surface to the nucleus.
4. The endothelial cell's machinery begins to produce new molecules including enzymes. These enzymes dissolve tiny holes in the sheath-like covering (basement membrane) surrounding all existing blood vessels.
5. The endothelial cells begin to divide (proliferate) and migrate out through the dissolved holes of the existing vessel towards the diseased tissue (tumor).
6. Specialized molecules called adhesion molecules called integrins (avb3, avb5) serve as grappling hooks to help pull the sprouting new blood vessel sprout forward.
7. Additional enzymes (matrix metalloproteinases, or MMP) are produced to dissolve the tissue in front of the sprouting vessel tip in order to accommodate it. As the vessel extends, the tissue is remolded around the vessel.
8. Sprouting endothelial cells roll up to form a blood vessel tube.
9. Individual blood vessel tubes connect to form blood vessel loops that can circulate blood.
10. Finally, newly formed blood vessel tubes are stabilized by specialized muscle cells (smooth muscle cells, pericytes) that provide structural support. Blood flow then begins.

Angiogenesis Facts & Figures

- Blood vessels are comprised of cells called endothelial cells. The total surface area covered by these cells in an adult is 1000 m2 -- roughly the size of a tennis court.

- If all the blood vessels in the body were lined up end-to-end, they would form a line that could

Seminal Papers

First Description of Tumor Vascularization:
Goldman E. The growth of malignant disease in man and the lower animals with special reference to the vascular system. *Lancet* 1907; ii: 1236-1240.

Seminal Hypothesis:
Folkman J. Tumor angiogenesis: therapeutic implications. *New England Journal of Medicine* 1971; 285: 1182-1186.

Early Discoveries:
Folkman J, Merler E, Abernathy C, Williams G. Isolation of a tumor factor responsible for angiogenesis. *Journal of Experimental Medicine* 1971; 133:(2): 275-288.

Gimbrone MA, Leapman S, Cotran RS, Folkman J. Tumor dormancy in vivo by prevention of neovascularization. *Journal of Experimental Medicine* 1972; 136 (2): 261-276.

Folkman J, Hochberg M. Self-regulation of growth in three dimensions. *Journal of Experimental Medicine* 1973; 138(4): 745-753.

Gimbrone MA, Cotran RS, Leapman SB, Folkman J. Tumor growth and neovascularization: an experimental model using the rabbit cornea. *Journal of the National Cancer Institute* 1974; 52(2): 413-427.

Orlidge A, D'Amore PA. Inhibition of capillary endothelial cells by pericytes and smooth muscle cells. *Journal of Cell Biology* 1987; 105: 1455-1462.

Leung DW, Cachianes G, Kuang WJ, Goeddel DV, Ferrara N. Vascular endothelial growth factor is a secreted angiogenic mitogen. *Science* 1989; 246(4935): 1306-1309.

Plouet J, Schilling J, Gospodarowicz D. Isolation and characterization of a newly identified endothelial cell mitogen produced by AtT-20 cells. *EMBO Journal* 1989; 8(12): 3801-3806.

First Angiogenesis Growth Factor:
Shing Y, Folkman J, Sullivan R, Butterfield C, Murray J, Klagsbrun M. Heparin affinity: purification of a tumor-derived capillary endothelial cell growth factor. *Science* 1984; 223 (4642): 1296-1299.

First Angiogenesis Inhibitor:
Brem H, Folkman J. Inhibition of tumor angiogenesis mediated by cartilage. *Journal of Experimental Medicine* 1975; 141:

First Clinical "Proof of Concept" of Antiangiogenic Therapy for Tumors:
White CW, Sondheimer HM, Crouch EC, Wilson H, Fan LL. Treatment of pulmonary hemangiomatosis with recombinant interferon alfa-2a. *New England Journal of Medicine* 1989; 320(18): 1197-1200.

Folkman J. Successful treatment of an angiogenic disease. *New England Journal of Medicine* 1989; 320(18): 1211-1212.

First Clinical "Proof of Concept" of Pro-angiogenic Therapy for the Ischemic Heart:
Schumacher B, Pecher P, von Specht BU, Stegmann T. Induction of neoangiogenesis in ischemic myocardium by human growth factors: first clinical results of a new treatment of coronary heart disease. Circulation 1998;97(7):645-650.

2004: A pivotal phase 3 trial published in the *New England Journal of Medicine* shows that the addition of bevacizumab (Avastin), an anti-VEGF monoclonal antibody, to chemotherapy significantly improves survival in patients with metastatic colorectal cancer.

2004: Bevacizumab is FDA approved for the treatment of advanced colorectal cancer. At the time of bevacizumab's approval, FDA Commissioner Mark McClellan declares antiangiogenic therapy "the fourth modality for cancer treatment."

2004: Pegaptanib (Macugen), an anti-VEGF aptamer, becomes the first anti-VEGF drug to be FDA approved for the treatment of age-related macular degeneration.

2004: Erlotinib (Tarceva), a small molecule inhibitor of EGFR tyrosine receptor kinase, receives FDA approval for treatment of non-small cell lung cancer (NSCLC).

2005: Endostatin (Endostar), an agent that inhibits metastasis and angiogenesis by downregulating multiple proangiogenic growth factors, is approved in China for the treatment of advanced lung cancer.

2005: Sorafenib (Nexavar), a multi-tyrosine kinase inhibitor, demonstrates significantly longer progression-free survival vs. placebo in patients with advanced renal cancer in a randomized phase 3 trial.

2005: Sorafenib is FDA approved as second-line therapy for advanced renal cancer.

2005: Lenalidomide (Revlimid), and agent with both immumomodulatory and antiangiogenic properties, is FDA approved for treatment of myelodysplastic syndrome.

2006: Sunitinib (Sutent), a multi-tyrosine kinase inhibitor, receives FDA approval as first-line therapy for advanced renal cancer and gastrointestinal stromal tumor (GIST).

2006: Ranibizumab (Lucentis), a fragment of the bevacizumab molecule, is FDA approved for the treatment age-related macular degeneration.

2006: Bevacizumab in combination with paclitaxel and carboplatin is shown to significantly improve progression-free survival, overall survival, and response rates in treatment-naïve patients with advanced NSCLC. This is the first time an antiangiogenic agent plus chemotherapy has been shown to prolong survival in NSCLC patients.

2007: Results from a randomized phase 3 trial published in the *New England Journal of Medicine* show a significant survival benefit for sorafenib vs. placebo in patients with advanced renal cancer who fail first-line therapy.

2007: Temsirolimus (Torisel), an inhibitor of mTOR, is approved for the treatment of advanced renal cancer after a pivotal phase 3 trial published in the *New England Journal of Medicine* shows significantly improved progression-free survival in previously untreated mRCC patients with poor prognosis.

2007: Results from a randomized phase 3 trial published in the *New England Journal of Medicine* show that sunitinib doubles progression-free survival in previously untreated patients with metastatic renal cancer.

2007: Results announced at ASCO 2007 from a randomized phase 3 study show that sorafenib extends overall survival by 44% vs. placebo in patients with advanced liver cancer. Based on these findings, in November the FDA approves sorafenib to treat unresectable advanced hepatocellular carcinoma. Sorafenib is the first systemic agent to show efficacy for advanced liver cancer.

2008: Angiogenesis pioneer Dr. Judah Folkman passes away suddenly on January 14 while traveling to a conference. At the time of Dr. Folkman's death, an estimated 1.2 million patients had been treated with antiangiogenic therapy, a concept he first conceived of almost 4 decades prior. Dr. Folkman is widely recognized as one of the most important figures in modern medicine.

Angiogenesis Inhibitors for Cancer

In the U.S., there are currently eight approved anti-cancer therapies with recognized antiangiogenic properties in oncology. These agents, which interrupt critical cell signaling pathways involved in tumor angiogenesis and growth, comprise two primary categories: **1) monoclonal antibodies** directed against specific proangiogenic growth factors and/or their receptors; and **2) small molecule tyrosine kinase inhibitors** (TKIs) of multiple proangiogenic growth factor receptors; **3) Inhibitors of mTOR** (mammalian target of rapamycin). In addition, at least two other approved angiogenic agents may indirectly inhibit angiogenesis through mechanisms that are not completely understood. Finally, in the field of dermatology, there are several agents used for neoplasms of the skin.

Monoclonal Antibody Therapies

Four monoclonal antibody therapies are approved to treat several tumor types: Bevacizumab (Avastin®), cetuximab (Erbitux®), panitumumab (Vectibix™), and trastuzumab (Herceptin®).

Bevacizumab (Avastin)	***Description***
Genentech	A humanized monoclonal antibody that binds biologically active forms of vascular endothelial growth factor (VEGF) and prevents its interaction with VEGF receptors (VEGFR-1 and VEGFR-2), thereby inhibiting endothelial cell proliferation and angiogenesis.
	Approved indications
	Metastatic colorectal cancer (mCRC), non-small cell lung cancer (NSCLC), advanced breast cancer. • In combination with 5-FU-based chemotherapy as first-line and second-line treatment of mCRC. • In combination with carboplatin and paclitaxel as first-line treatment of patients with unresectable, locally advanced, recurrent or metastatic non-squamous NSCLC. • In combination with paclitaxel as first-line treatment in patients with locally recurrent or metastatic breast cancer.
Cetuximab (Erbitux)	***Description***
Bristol-Myers Squibb ImClone	A chimeric IgG1 monoclonal antibody that binds the extracellular domain of epidermal growth factor receptor (EGFR), preventing ligand binding and activation of the receptor. This blocks downstream signaling of EGFR, inhibiting cell proliferation and angiogenesis, among other effects.

Small Molecule Tyrosine Kinase Inhibitors (TKIs)

Three TKIs are currently approved as anticancer therapies: Erlotinib (Tarceva®), sorafenib (Nexavar®), and sunitinib (Sutent®).

Erlotinib (Tarveca)	***Description***
Genentech OSI Roche	Small molecule TK inhibitor of EGFR.
	Approved indications
	NSCLC, pancreatic cancer. • Monotherapy for locally advanced or metastatic NSCLC in patients who have failed at least one prior chemotherapy regimen. • In combination with gemcitabine as first-line treatment of locally advanced, unresectable or metastatic pancreatic cancer.
Sorafenib (Nexavar)	***Description***
Bayer Onyx	Small molecule TK inhibitor of of VEGFR-1, VEGFR-2, VEGFR-3, PDGFR-ß, and Raf-1.
	Approved indications:
	Advanced renal cell carcinoma, advanced hepatocellular carcinoma. • Treatment of unresectable hepatocellular carcinoma.
Sunitinib (Sutent)	***Description***
Pfizer	Small molecule TK inhibitor of VEGFR-1, VEGFR-2, VEGFR-3, PDGFR- ß, and RET.
	Approved indications:
	Advanced renal cell carcinoma, GIST. • Treatment of gastrointestinal stromal tumor (GIST) after disease progression on or intolerance to imatinib mesylate.

Inhibitors of mTOR

One mTOR inhibitor, temsirolimus (Torisel), is currently approved as anti-cancer

related Kaposi's sarcoma (KS). Retinoids, derivates of vitamin A, are antiangiogenic via downregulation of VEGF.

- Imiquimod (Aldara 5% cream, Graceway) is a Toll-Like Receptor 7 aganist which is an immune response modifier that exerts antiangiogenic activity through local upregulation of interferons and interleukins, downregulation of FGF-2 and MMP-9, and induction of endothelial apoptosis. Imiquimod is indicated for both benign neoplasms (genital warts) and for malignant skin cancers (actinic keratosis and basal cell carcinoma).
- Interferon alfa (Intron and Roferon) is a pharmacologic version of an endogenous cytokine with antiangiogenic activity that is administered systemically. Interferon has been used off-label to treat hemangiomas and giant cell tumors in pediatric patients.
- Polyphenon E (Veregen 15% ointment, Bradley/MediGene) is a defined composition of polyphenolic kunecatechins extracted from green tea leaves. The major green tea catechins, epigallocatechin-3 (EGCG), inhibits VEGF expression. Polyphenon E topical ointment indicated for genital warts.

Inhibitors for Eye Disease

Angiogenesis in the eye underlies the major causes of blindness in both developed and developing nations: exudative age-related macular degeneration (AMD), proliferative diabetic retinopathy (PDR), diabetic macular edema (DME), neovascular glaucoma, corneal neovascularization (trachoma), and pterygium. Presently approved anti-angiogenic therapies for ophthalmic conditions are biologic agents that inhibit VEGF. There are currently two approved antiangiogenic therapies for ophthalmic diseases: an anti-VEGF aptamer (pegaptanib, Macugen); and a Fab fragment of a monoclonal antibody directed against VEGF-A (ranibizumab, Lucentis).

Monoclonal Antibody Therapy

One monoclonal antibody therapy is approved to treat Age-related macular degeneration (AMD), a progessive eye diease that results in loss of central vision, and is the leading cause of severe vision loss in adults over the age of 65. The wet form of AMD accounts for 10% of cases, and is characterized by the abnormal growth of new blood vessels, which leak fluid and blood, inducing scare formation and destroying vision.

Ranibizumab	***Description***
Lucentis	A recombinant humanized IgG1 kappa monoclonal antibody fragment that binds vascular endothelial growth factor-A (VEGF-A) and cleavage products, and prevents their interaction with VEGF receptors (VEGFR-1 and VEGFR-2), thereby inhibiting endothelial cell proliferation, angiogenesis, and vasular leakage in the retina and choroidal layers.
	Approved indications:
	Neovascular (wet) age-related macular degeneration • Administered by intravitreal injection.

Aptamer

Pegaptanib	***Description***
OSI Eyetech Pfizer	A pegylated modified oligonucleotide (aptamer) which adopts a three dimentional conformation that enables it to bind to extracellular VEGF, thereby inhibiting its binding to VEGF receptors and suppressing pathological neovascularization.